The Newsletter of the Australian and New Zealand Society of Paediatric Dentistry

Synopses

OCTOBER 2009, ISSUE 43

www.anzspd.org.au

Rickets disease in children and adolescents: a narrative review

Charmaine Hall BDSc, second year post-graduate student in DCD program in Paediatric Dentistry
Melbourne Dental School, University of Melbourne
Email: c.hall4@pgrad.unimelb.edu.au

Abstract

Rickets disease in children and adolescents is re-appearing in the population, especially amongst recent immigrants. Rickets disease is caused by vitamin D deficiency and has an environmental and/or genetic aetiology. The disease is characterised by softened bones that are prone to deformity and fractures. Children with rickets disease have been reported to have several dental anomalies that may lead to premature loss of the primary dentition, defective enamel and malocclusion of the permanent dentition. It is imperative that dental clinicians be alert to this condition as the systemic features may be mild with dental manifestations as the first presenting signs.

Introduction

Rickets is caused by vitamin D deficiency and it is characterised by soft pliable bones that become deformed due to defective mineralisation of osteoid. Vitamin D deficiency is also characterised by low circulating concentrations of calcium and an increased serum alkaline phosphatase activity[1,2]. This article will briefly cover the role of vitamin D in the body, the pathogenesis of rickets disease and also the systemic and dental manifestations of rickets disease. The management of dental complications will also be discussed.

Sources of vitamin D

There are intrinsic and extrinsic sources of vitamin D. Vitamin D refers to both D3, cholicalciferol (produced in the skin) and D2, ergocalciferol (the form in food). Vitamin D (calciol) is really a hormone; it is only under conditions of inadequate exposure to sunlight that dietary intake is required[1]. Vitamin D is the only vitamin that is not usually required in the diet. It is mostly produced by the action of ultraviolet light on provitamins, ergosterol in plants and 7-dehydrocholesterol in animals which is synthesised in the liver and is found in the skin. Extrinsic sources of vitamin D include foods such as fish, liver and there are small amounts in eggs. Some dairy products (milk, margarines) are also fortified with vitamin D3.

CONTINUED ON PAGE 4...

THIS ISSUE

President's Report

Nina Vasan

PHOTOS OF WORLD VISION SPONSOR VISIT IN ARUSHA

Another year racing by! I was not sure what to write for this edition, when I received an email yesterday from Dr Kylie Pearce to let me know about two distinguished people from the Sunshine Coast that had died tragically in a plane trip across Paupa New Guinea. One of whom was Dr June Canavan, a local sports medicine physician who had taken up a quest to conquer 5 treks all starting with "K" to raise money for the School of St Jude in Tanzania.

Dr Canavan's project was called Klocking up the K's, and aimed to raise much needed funds for the School of St Jude, run by an Australian woman in Tanzania. A friend, Keith Gracie had joined her for the Kokoda leg of the quest. Tragically Dr June and Keith were killed before their dream was realised. Kylie is due to climb Mt Kilamanjaro in September (which she would have completed when this newsletter reaches you). Kylie was allowed to climb on behalf of the Klocking up the K's charity and help achieve the $50,000 target that Dr June had aimed for. Kylie's email to me stated "my views are that children can only reach their full potential if they are given every possible opportunity to succeed. Without basic education these children would never know what life could offer. Dr Canavan has inspired me to contribute to this wonderful cause." I'm sure this is a sentiment felt by all of us.

There is more information regarding this worthy cause on www.klockinguptheks.com.au and later in this edition of Synopses you can find out how you can contribute to this cause.

One of my patients, Haydn and his family are currently in Arusha, Africa visiting their World Vision sponsor, Ashraf. They send me email updates of their trip, encounters and the people they meet. I have attached some photographs, in particular of the school that Ashraf attends. Part of the email Haydn's mother sent me reads …

"Ashraf has one brother, Amecci, who is 9 (Ashraf is 7). Their aunty also lives with them. His Nan lives in the same village. They live in a one roomed 'shack', with no doors in the doorways (or windows) made of bricks made by the local men of mud. They have one bicycle and they use that to fetch water which is 1.5km away. They have no electricity and Ashraf and his brother walk the 3km to school and back (twice a day, as the kids come home for lunch from 12-2pm then back to school at 2pm till 5pm)."

As you can see the school is a far cry from the ones that our children attend in New Zealand and Australia. Helping to educate these children is a great way to give them a better 'kick start' to life and help the entire community for years to come.

Is your adolescent patient taking Methamphetamines?

In NZ, there is an increasing number of young people taking recreational methamphetamines. One of my now 15 year old patients, whom I had been seeing regularly since he was 4 years old, had recently started to take the drug 'P'. His oral hygiene levels had declined dramatically (last brushed two weeks prior), there was generalised demineralisation at the gingival margin of most teeth, a dry mouth and a number of new carious lesions developing. His mother had informed me prior that he was taking a number of recreational drugs, in particular 'P'. It is difficult to see a child patient who was bright and bubbly and full of life, become a teenager who is withdrawn and depressed. Naturally, this would be extremely heartbreaking for the parents. As methamphetamine usage becomes more prevalent, I needed to find out more about this destructive drug.

Some basic information that healthcare providers should know:

- Amphetamines comprise a closely related family of drugs with psychoactive properties. They include amphetamines, methamphetamine, dexamphetamine and methylphenidate (Ritalin). Methamphetamine is the most potent form of amphetamine available.

- There are estimated to be 26 million amphetamine and methamphetamine users worldwide. Reported use of amphetamines is highest in the UK, Australia and New Zealand.
- Methamphetamine is a synthetic drug that is sold under different street names some include: 'P', 'speed', 'meth', 'ice', 'crack', 'glass', 'base'. The drug sells for about $100-180 per gram, and more pure forms for about $1000 per gram. Common abused doses are 100-1000mg daily and up to 5000mg in chronic binge use.
- Methamphetamine can be taken by smoking/inhalation, snorting, orally, intravenously or rectally
- Methamphetamine stimulates the central and peripheral sympathetic nervous systems by increasing synaptic concentrations of the excitatory neurotransmitters dopamine, noradrenaline and serotonin. Increased noradrenaline concentrations contribute to the anorectic (appetite suppressing), locomotor and sympathomimetic effects, while increased dopamine concentrations stimulate locomotor effects, psychosis and perception disturbances; and serotonin is responsible for delusions and psychosis.
  - ***CNS effects*** – euphoria; increased well-being, confidence and physical activity; improved cognitive performance; suppression of appetite and need for sleep
  - ***PSNS effects*** – increased blood pressure, tachycardia, increased temperature. Large doses can cause cardiac arrhythmias
- Repeated and prolonged use leads to marked tolerance (decreased drug response), and increasing doses are required to maintain the same effects. Prolonged use results in marked psychological dependence.
- Long term effects include: psychosis (delirium, paranoia, tactile hallucinations); depression (suicide attempts are common); association with violence. Recent Australian data indicated that 12% of meth-amphetamine users had committed a violent crime in the preceding year.
- Treatment options are limited as there are no established pharmacotherapies, as there are with alcohol and opioids. Management largely relies on psychosocial interventions, and cognitive behaviour therapy. Most users become abstinent through self-management when they tire of their tumultuous lifestyle.

McAvoy BR. Journal of Primary Health Care: Vol 1 (3); Sept 2009

Dental Implications of methamphetamine abuse

Dental patients who abuse methamphetamine can present with poor oral hygiene, xerostomia, rampant caries ('Meth mouth'), and excessive tooth wear. Dental management of methamphetamine users requires obtaining a thorough medical history and performing a careful oral examination. The most important factor in treating the oral effects of methamphetamine is for the patient to stop using the drug. Continued abuse will make it difficult to increase salivary flow and hinder the patient's ability to improve nutrition and oral hygiene. Local anesthetics with vasoconstrictors should be used with care in patients taking methamphetamine because they may result in cardiac dysrhythmias, myocardial infarction, and cerebrovascular accidents. Thus, dental management of patients who use methamphetamine can be challenging. Dentists need to be aware of the clinical presentation and medical risks presented by these patients.

Hamamoto, D T; Rhodus, N L Oral Diseases. 15(1):27-37, January 2009.

Recent Dental Events: The IAPD conference was held in Munich (June 2009) and a number of our Members attended. The feedback was very positive. The highlight has to be, Dr Eduardo Alcaino being nominated and successful in becoming 'President Elect' for the IAPD. The process to becoming President Elect is extremely competitive and a number of factors are taken into account as well as recommendations from several dental organisations – it is by no means an easy task! This is a huge privilege and we congratulate Eduardo on a great achievement. All the very best Eduardo for this upcoming role!

The Upcoming ANZSPD Conference

This is my last opportunity to give a BIG 'plug' for the upcoming ANZSPD Conference to be held in Queenstown 28-30 March 2010. The programme is looking fabulous with Dr Katie Ayers and the organising committee bringing together a range of speakers to discuss current and interesting topics. As well as some unforgettable social events.

Check out the website

www.conference.co.nz/anzspd2010 for more details. For those who have never been to Queenstown, this is a fantastic time to bring the kids, do a trip around the beautiful South Island, or even pack in a tramp through the Milford Track. Check out the photos below! I would also like to take this opportunity to thank Colgate for once again being the Principal Sponsor for the conference. ANZSPD is grateful for the continual support received at all levels from Colgate.

Have a fantastic Christmas and well deserved break with your families. See you in 2010!

Nina

CONTINUED FROM PAGE 1...

Pathogenesis of Rickets disease

Rickets disease is caused by either (or both) environmental and genetic factors. Rickets is a result of mutations of genes involved in the vitamin D pathway or secondary to nutritional deficiency[4,5]. Vitamin D regulates the transcription of genes and gene expression of proteins involved in the formation of mineralised tissues[5,6]. Deficient vitamin D therefore results in defective mineralisation of bones and teeth.

Vitamin D3 plays an important role in calcium and phosphate homeostasis[4]. In the intestinal epithelial cells vitamin D3 induces gene expression affecting calcium metabolism. The active metabolite of vitamin D is $1,25\text{-}(OH)_2D_3$ which increases the absorption of calcium and phosphate from the gut via active transport by calcium-binding proteins. Together with parathyroid hormone, vitamin D stimulates bone resorption by osteoclasts resulting in increased serum calcium and phosphate concentrations. Low $1,25\text{-}(OH)_2D_3$ causes low calcium and phosphate availability and reduced osteoblast function, resulting in rickets disease in children[1].

Hereditary defects in vitamin D metabolism

The three main hereditary defects in vitamin D metabolism are:

1. Vitamin D-dependent rickets type I (VDDRI)
2. Vitamin D-dependent rickets type II (VDDRII)
3. Familial hypophosphataemic rickets aka 'vitamin D resistant rickets'

Vitamin D-dependent rickets type I (VDDRI)

Vitamin D-dependent rickets type I is a rare autosomal recessive disease that is also known as pseudo-vitamin D-deficiency rickets[7]. The disease is characterised by the deficiency or absence of the renal enzyme 1–α–hydroxylase that is responsible for the bioactivation of vitamin D[7]. Vitamin D-dependent rickets type I appears with high frequency in the Canadian French population of Quebec and manifests in the first year of life[7]. Distinguishing clinical manifestations of VDDRI include growth failure, short stature, skeletal abnormalities, genu valgum ('knock-knees'), rachitic rosary, open fontanelles, pathologic fractures, muscle weakness and convulsions. Radiologic findings usually include generalised osteopenia, growth failure, arched or curved legs and bone fractures[7]. There appear to be no published reviews detailing the intraoral manifestations of VDDRI. A recent case report of VDDRI in a 10-year old Venezuelan girl noted enamel hypoplasia, defective dentine, large quadrangular pulp chambers, short roots, generalised periodontal disease and maxillary growth deficiency as the main intraoral findings[7].

Vitamin D-dependent rickets type II (VDDRII)

Vitamin D-dependent rickets type II is an autosomal recessive form of rickets caused by an abnormality in the vitamin D receptor, producing target organ resistance to 1,25(OH) vitamin D[7]. The disease is also known as hypocalcaemic vitamin-D dependent rickets.

Familial hypophosphataemic rickets (FHVDDR) or 'Vitamin D resistant rickets'

Familial hypophosphataemic vitamin-D dependent rickets (FHVDDR) is the third type of hereditary rickets which represents an X-linked dominant trait associated with mutations in the PHEX gene (phosphate regulating gene)[7]. Recently, other modes of transmission have been reported: autosomal recessive for the mutation on the gene encoding dentine matrix protein 1 and autosomal dominant transmission for the gene encoding fibroblast growth factor 23[8]. Familial hypophosphatemic rickets belongs to a larger family of vitamin-D resistant rickets which occurs in 1 out of every 20,000 births and affects males more severely. This type of infantile rickets is the most common form[8].

The principal aetiology is a mutation of the PHEX gene located on the distal part of the short arm of the X chromosome[8]. This gene encodes PHEX, an enzyme which has the capacity to inactivate indirectly the hypophosphatemic circulating factors, such as fibroblastic growth factor 23. Alteration in the PHEX gene also results in impaired phosphate transport with reduced phosphate reabsorption in the renal proximal tubules of the glomerular system of filtration as well as the intestine[8]. This causes hyperphosphaturia and hypophosphatemia as well as impaired 1–α–hydroxylation of 25–hydroxyvitamin D. Additionally, PHEX might ensure a specific interaction with matrix extracellular phosphoglycoprotein of bone and dentine matrix and could protect it from enzymatic cleavage within the matrix[8]. When PHEX is mutated, a motif is released by this cleavage which may hinder mineralisation. The consequential accumulation of non-mineralised matrix and slightly mineralised bone constitutes the origin of the clinical signs of the disease[8].

Environmental risk factors for vitamin D deficiency

The normal level for vitamin D in children is 50-160 nmol/L. Vitamin D deficiency is defined as levels < 25nmol/L and insufficiency as levels 25-50 nmol/L[3]. The peak incidence of vitamin D deficiency is between 3 and 18 months of age[9]. Individually or in combination, common causes of vitamin D deficiency are reduced exposure to sunlight, poor dietary intake, malabsorption or abnormal metabolism of vitamin D caused by liver disease (deficient 25–hydroxylation) or renal failure (1–α–hydroxylase deficiency)[1,2]. Additionally, some medications such as anticonvulsants and isonizid can contribute to low vitamin D levels in some patients because they induce liver enzymes which increase the metabolism of vitamin D thereby reducing serum levels[3].

Reduced exposure to sunlight commonly occurs in institutionalised

elderly persons and immigrants from the Middle East or Indian subcontinent who wear traditional dress[1]. People with darker coloured skins may need to spend up to 6 times as long in the sun as someone with a fairer complexion to make an adequate quantity of vitamin D, which may be up to 5 hours a day in winter (with arms and face exposed)[10]. The amount of sunlight required for adequate vitamin D in children is not known[11].

Vitamin D deficiency caused by malnutrition is uncommon[3]. Most Western diets contain sufficient vitamin D, however strict vegetarian or lactovegetarian diets have inadequate vitamin D content and in the long term may result in insufficiency[1]. Malabsorption of vitamin D may be caused by coeliac disease, Crohn's disease, pancreatic insufficiency, inadequate bile-salt secretion and non-tropical sprue[1].

In Australia, risk factors for low vitamin D include dark skin colour, reduced sun exposure (especially where skin in covered up/time inside), duration since immigration (shorter duration may imply more time indoors) and breast feeding with other risk factors present (skin colour, low maternal vitamin D levels, time indoors)[3]. It has been reported that up to 90% of the African population in Melbourne, Australia have low vitamin D[3]. The aforementioned factors may have contributed to this high percentage.

Systemic and dental manifestations of rickets disease

Mineralised tissue deformities have been reported with all types of rickets. The currently accepted interpretation of such bone deformities is the combination of tissue hypomineralization and mechanical disorders induced by muscle function impairment[6]. The variation in clinical signs is due to several factors: family history, the degree of hypophosphataemia and the age of the patient at the start of systemic treatment[8]. Prominent systemic clinical symptoms are deformities in the limbs, gait disturbance, dwarfism, familial occurrence, bowed legs and knock-knees[12]. The latter two major signs of the disease become obvious with the onset of the child's first steps which are complicated by this dysmorphism[8]. Radiographic examination reveals osseous structures with less dense trabeculations and the lamina dura is often absent.

Dental manifestations include[8,12,13]:

- Dental anomalies and delayed eruption of both the primary and permanent dentitions,
- Enamel of normal quality but thinner (enamel hypoplasia) with long internal cracks,
- Dentine with many calcospherites separated by irregular zones of interglobular dentine[14] ,
- Very large pulp chambers which may be taurodont. Pulp horns are especially high in primary teeth and can extend up to the dentine-enamel junction. Taurodontism of permanent molar teeth may occur in more severely affected male patients[15].
- Spontaneous multiple dental abscesses, acute or chronic, with no history of trauma or dental caries is frequently found in patients with hypophosphataemic rickets (FHVDDR). This occurs mostly in the primary dentition and follows the natural sequence of eruption of these teeth.
- Ectopically erupted permanent canines[16].

Progression of bacteria is facilitated by the prominent pulp horns, cracks in enamel and dentine and the wearing of thin enamel. The propensity for occurrence of multiple abscesses in patients with VDDRI and VDDRII is not known[7]. Patients with hypophosphataemic vitamin-D-resistant rickets often have deficiency in the anterior cranial base length, ramus height and cranial base angle. A class III skeletal relationship is also observed frequently in affected patients[16].

Molecular mechanisms of enamel and dentine dysplasia in rickets affected patients

Little is known about the molecular mechanisms involved in enamel and dentine dysplasias which are commonly found in affected patients. The mineralisation defects may result from the abnormal environment of the ameloblasts and odontoblasts in patients with an altered PHEX gene. Patients with this gene have low phosphate concentrations in extra-cellular fluids and low serum concentrations of 1,25 di-hydroxyvitamin D[17]. The localisation of a PHEX mRNA expression in osteoblasts and odontoblasts suggests a local effect of the deficient production of the PHEX-derived protein[17].

Early management of the vitamin D deficiency is imperative to minimise the effects on the dentition. Early dosages of 1,25–$(OH)_2D_3$ may exert a direct effect on the odontoblasts and ameloblasts as these cells express the vitamin D receptor and respond *in vitro* to this vitamin[17]. Patients whose phosphate and vitamin D metabolism are normalised earlier will have a better dental status[17].

Possible cross-talk between vitamin D, vitamin D receptor and the Msx2 homeobox gene (genes responsible for mineralised tissue formation) is a suggested influencing factor in dental tissue dysplasias[6]. One *in vivo* study on rats suggested that specific dysplasia of rachitic enamel (decrease of intraprismatic enamel) is secondary to vitamin D dysregulation of amelogenin expression. The 1,25 $(OH)_2D_3$ up-regulates the transcription of rat amelogenin gene and can therefore alter amelogenesis[5]. The inter-relationship and regulatory mechanisms of vitamin D and amelogenin expression are still unclear[4].

Dental management of the patient with rickets disease

Early diagnosis of these conditions is necessary to prevent major dental abnormalities and bone deformities. A multidisciplinary approach to patient management in a paediatric hospital is imperative for children

with clinical rickets or abnormal serum calcium[3]. Management involves referral to a paediatrician, nutritionist, orthopaediac surgeon, specialist in metabolic medicine and a geneticist.

After a thorough clinical examination, essential investigations include radiographs of limbs and the chest and serology tests. Radiographs may reveal widened, cupped or frayed metaphyses, osteopenia, rib flaring or fractures[2]. Serology tests are required to assess calcium, phosphate, alkaline phosphatase and vitamin D serum levels. These tests enable the clinician to diagnose rickets and to monitor treatment. Clinical photography is also useful to monitor bone deformity[3]. Screening of infants who are at risk of low vitamin D is ideal but not always feasible. Children who have recently immigrated to Melbourne, Australia are seen at the Royal Children's Hospital and will be initially assessed for their vitamin D levels[3]. If clinical rickets is present, additional investigations are required; parathyroid hormone function, renal function and a wrist x-ray[3].

Early treatment of the vitamin D insufficiency or deficiency is important to minimise the effects of the disorder on the bone and teeth. Additional normalisation of calcium and phosphate levels also restores (to a large extent) normal bone morphology and muscle function of rachitic patients[6]. The Royal Children's Hospital of Melbourne uses cholecalciferol (D3) 100,000 IU/ml in olive oil[3]. Breast - fed infants of mothers at risk of low vitamin D are encouraged to take 400IU Vitamin D daily (Pente–Vite™ 0.45ml oral daily)[3]. After vitamin D is given, the child's dietary intake of calcium must also be sufficient. Calcium supplements may be needed in patients with low dietary intake and should be administered under the supervision of a nutritionist[3].

A paediatric dentist is required to co-ordinate the dental team of specialists who manage patients with severe dental effects of rickets. Appropriate consultations with dental hygienists, prosthodontists and orthodontists may be warranted. A pertinent patient history and thorough clinical dental examination are essential to rule out similar metabolic and developmental dental structural disorders. Dental radiographs and study models aid in assessment of the dental defects and treatment planning.

A treatment plan should focus on prevention of problems associated with deep invisible dentine-enamel cracks which are subject to exposure from gradual tooth wear. To prevent adverse pulpal outcomes, periodic examinations, maintenance of good oral hygiene[12], sealing of cracks and grooves of both primary and permanent molars using a resin-based sealant[8], topical application of fluoride varnish[8,12], resin composite covering the anterior teeth and stainless steel crowns (SSC's) to cover the crowns of primary molars have all been advocated[8].

The literature shows different clinical approaches to the management of affected rachitic patients. For the young patient, prophylactic coverage of the molar teeth with SSC's has been advised[18]. As the placement of SSC's using conventional techniques requires removal of relatively large amounts of tooth structure, the risk of pulp exposure is high. Instead, a conservative approach of non-removal of tooth structure has been suggested[18]. This approach employs the use of separating elastics prior to placing a SSC[19]. In severe cases, it may be necessary to protect the occlusal surfaces of partially erupted molars with composite resins prior to insertion of metal crowns which is performed when the teeth are fully erupted[19]. The SSC may be converted to full gold or porcelain crowns when adulthood is reached[19].

Given the propensity for periapical abscess formation (especially the primary molars) in hypophosphataemic rickets, some authors have recommended an aggressive interventive regimen of prophylactic formocresol pulpotomies and SSC's[7]. However there is clinical evidence that both formocresol and ferric sulphate pulpotomies fail in these patients[20]. A controversial approach of pulpectomies and SSC's has also been advocated, but failure has been noted[21]. Early loss of abscessed primary teeth appears to be common, so managing the extractions and space loss for these patients is important[19].

For the older patient, there is very little in the literature on the management and success of endodontic treatment therapy in the permanent dentition. Goodman et al (1998) followed up 17 patients from 1973 to 1997, with 50% of the cases having abscesses in the permanent dentition (mainly associated with maxillary or mandibular incisors). Three of the root canal treated incisors developed pulp necrosis and periapical lesions without carious defects, trauma history or significant attrition. The loss of pulp vitality may have been the result of penetration of microorganisms through the micro-clefts in the enamel layer which were termed 'enamel infractures' by the authors[15]. A recent case from Melbourne reported successful endodontic treatment on a 16-year old boy who had six permanent anterior teeth which had spontaneously become non-vital and necrotic[22]. The affected incisors had bizarre root morphology which posed an endodontic challenge. The authors suggested that an acrylic night-splint be worn by affected patients to reduce natural attrition which may otherwise predispose such vulnerable teeth to pulp exposure and subsequent abscess formation[22].

In a recent report of an affected case, all primary molars were sealed with a resin-based sealant rather than placing SSCs because the technique is less aggressive, not all pulp tissue in affected teeth is infected, and iatrogenic pulp infection may occur during the crown preparation[8]. The aim of the fissure sealants was to create a mechanical barrier to bacterial invasion by sealing the cracks. A self-etching bonding system was used because of its clinical simplicity (absence of rinsing, decreased operative time) and less aggressive etching of the enamel which avoids damaging cracks that are already present[8]. The authors recommended that the sealants be reviewed regularly and repaired as loss due to attrition of the resin composite or mechanical loss is anticipated. The use of adhesive

dentistry may prevent abscesses in affected teeth and can be implemented in a child at an age where co-operation to treatment is not always guaranteed[8].

Conclusion

Rickets is becoming more prevalent in Western societies with increased immigration of at – risk individuals. The dental clinician must be able to recognise the dental side effects and know when to refer to appropriate specialists to manage these patients effectively.

Bibliography

1. Baynes JW, Dominiczak MH. Medical Biochemistry 2nd edition Elsevier Mosby 2005.
2. www.rch.org.au/intranet/fracp_resources (last accessed August 1, 2009)
3. www.rch.org.au/immigranthealth/resources (last accessed August 1, 2009)
4. Papagerakis P, MacDougall M, Berdal A. Differential epithelial and mesenchymal regulation of tooth-specific matrix proteins expression by 1,25-dihydroxyvitamin D3 in vivo. Connc Tiss Res 2002; 43: 372-75.
5. Papagerakis P, Hotton D, Lezot F, Brookes S, Bonass W, Robinson C, Forest N, Berdal A. Evidence for regulation of amelogenin gene expression by 1,25-dihydroxyvitamin D3 in vivo. J Cell Biochem 1999; 76: 194-205.
6. Lézot F, Descroix V, Mesbah M, Hotton D, Blin C, Papagerakis P, Mauro N, Kato S, MacDougall M, Sharpe P, Berdal A. Cross-talk between MSX/Dlx homeobox genes and vitamin D during tooth mineralisation. Connec Tiss Res 2002; 43: 509-14.
7. Zambrano M, Nikitakis NG, Carmen Sanchez-Quevedo M, Sauk JJ, Sedan H, Rivera H. Oral and dental manifestations of vitamin-D dependent rickets type I: Report of a paediatric case. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003; 95: 705-9.
8. Douyere D, Joseph C, Gaucher C, Chaussain C, Courson F. Familial hypophosphatemic vitamin-D resistance rickets – prevention of spontaneous dental abscesses on primary teeth: a case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009: 107: 525-30.
9. Wagner CL, Greer FR. Prevention of rickets and vitamin D deficiency in Infants, Children and Adolescents. Pediatrics 2008; 122: 1142-52.
10. Clemens T, Adams J, Henderson S, Holick M. Increased skin pigment reduces the capacity of the skin to synthesise vitamin D3. Lancet 1982; 1: 74-6.
11. Munns C, Zacharin MR, Rodda CP, Batch JA, Morley R, Cranswick NE et al. Prevention and treatment of infant and childhood vitamin D deficiency in Australia and New Zealand: a consensus statement. Med J Aust. 2006; 185: 268-72.
12. Murayama T, Iwatsubo R, Akiyama S, Amano A, Morisaki I. Familial hypophosphatemic vitamin D-resistant rickets: dental findings and histologic study of teeth. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000; 90: 310-6.
13. Hillman G, Geurtsen W. Pathohistology of undecalcified primary teeth in vitamin D-resistant rickets: review and report of two cases. Oral Surg Oral med Oral Pathol Oral Radiol Endod 1996; 82(2): 218-24.
14. Seeto E, Seow WK. Scanning electron microscope analysis of dentine in vitamin D-resistant rickets-assessment of mineralisation and correlation with clinical findings. Paed Dent 1991;13: 43-8.
15. Goodman JR, Gelbier MJ, Bennett JH, Winter GB. Dental problems associated with hypophosphataemic vitamin D resistance rickets. Int J Paed Dent 1998; 8: 19-28.
16. Al-Jundi SH, Dabous IM, Al-Jamal GA. Craniofacial morphology in patients with hypophosphataemic vitamin-D-resistant rickets: a cephalometric study. J Oral Rehab. 2009: 36: 483-490.
17. Chaussain-Miller C, Sinding C, Wolikow M, Lasfargues J, Godeau G, Garabedian M. Dental abnormalities in patients with familial hypophosphatemic vitamin-D resistant rickets: prevention by early treatment with 1-hydroxyvitamin D. J Pediatr 2003; 142: 324-31.
18. Page LA. Vitamin D-resistant rickets: report of a case. NZ Dent J 2008; 104: 111-14.
19. Seow WK. Diagnosis and management of unusual dental abscesses in children. Aust Dent J 2003; 48: 156-68.
20. Coelho A, Marwues P, Canta JP. Case report: dental treatment of a child with hypophosphataemic rickets. Eur Arch Paed Dent 2007; 8(Suppl.1): 35-8.
21. Rakocz M, Keating J, Johnson R. Management of primary dentition in vitamin D-resistant rickets. J Oral Surg Oral Med Ora Pathol 1982; 54: 166-71.
22. Alexander S, Moloney L, Kilpatrick N. Endodontic management of a patient with x-linked hypophosphataemic rickets. Aust Endo J 2001; 27: 57-61.

ANZSPD (Victoria) Paediatric Oral Health Small Grants 2009

Applications are invited from interested individuals, groups, institutions or organisations for funding for small projects or items of equipment that will promote the oral health of children. Requests in writing should be made to the President, ANZSPD (Vic Branch) for funding of between $500 and $5000. The application should be no longer than one page including a clear aim, expected outcomes and how they meet the objectives of ANZSPD (Vic Branch) which are: 1. to study and promote the improved dental health of children; 2. to bring together individuals and entities from the various disciplines associated with paediatric dentistry for the purpose of furthering the objective above; 3. to provide opportunity for discussion of clinical methods and research based on the team approach; and 4. to do any other act or thing, ancillary or conducive to, and not inconsistent with the above. Applications should be sent by email in Word format to: Dr David Manton, President, ANZSPD (Vic Branch), djmanton@unimelb.edu.au

Applications will be considered by the ANZSPD (Vic Branch) committee whose decision will be final and no correspondence will be entered in to regarding the outcome of applications. Successful applicants will be expected to provide a report on the outcomes of their application for publication in Synopses.

Closing date for applications: Friday, 27 November 2009

Announcement of successful applicants will be made at the subsequent ANZSPD (Vic Branch) meeting.

Federal Secretary-Manager's Report

Alistair Devlin,
Secretary-Manager, ANZSPD Inc.

2010 A.N.Z.S.P.D. – Colgate Paediatric Dentistry Post-graduate Research Award

Through the generous financial support supplied by Colgate Oral Care, any post-graduate student currently enrolled in a paediatric dentistry Masters degree or Ph.D/Doctorate program, or any such student who has completed any of one of these programs since May 2007, is invited to compete for this award.

Competitors are required to present a 10 minute paper at the forthcoming 16th A.N.Z.S.P.D. Convention in Queenstown, New Zealand on a completed research project undertaken in their graduate program.

The prize is a single return economy airfare and a cash prize of AU$500.00 to assist in the payment for registration and accommodation to attend and present the same paper at an appropriate international paediatric dental meeting e.g. the 23rd International Association of Paediatric Dentistry Congress in Athens in June, 2011.

Please contact Alistair Devlin at devlins@iinet.net.au for details on the competition

Entries for the Award will close on 31st December 2009.

The A.N.Z.S.P.D. Grant

Preliminary notice is given of this new scheme which was adopted by the A.N.Z.S.P.D. Federal Council at it's meeting in March of this year. The scheme will provide one Grant per year to the value of AU$2,000. The Grant will be available to any A.N.Z.S.P.D. members for:

- an oral health initiative in Australia or New Zealand which may be an educational resource or a broad community initiative.
- a community research project directly related to child oral health.
- support for an oral health project in Asia, Oceania or the Pacific which might be for materials, instruments, books for a school, etc.

Applications for the Grant will be called for in the first edition of Synopses in 2010.

The A.N.Z.S.P.D. Annual General Meeting

Notice is hereby given to all A.N.Z.S.P.D. members of the Annual General Meeting of the Society. This will be held at the 16th A.N.Z.S.P.D. Convention in Queenstown, New Zealand in March 2010. Exact details of the time and venue of the meeting will be advertised in the Convention brochure and on the Convention website www.anzspd2010.com

Constitutional Amendment

At the Annual General Meeting as mentioned previously, the following amendment to the Constitution of A.N.Z.S.P.D. Inc. will be moved:

"That the A.N.Z.S.P.D. By Laws, Chapter 1, Section 1 be amended by adding a fourth category of membership: "(4) Honorary life members. In recognition of signal service to the Society, the Council may recommend the conferring of Honorary Life Membership on a suitably qualified full member."

The other three existing categories of membership are Full, Honorary and Associate. The new category will distinguish the difference between the current Honorary members, who are not necessarily dentists or perhaps not residing in Australia or New Zealand and the new fourth category which aims to recognise and honour the selfless contribution to the Society by any of its full members.

International Association of Paediatric Dentistry Individual Memberships

Just to reiterate that from 2010, A.N.Z.S.P.D. Inc. will no longer act as an agent in facilitating Individual Memberships of I.A.P.D. A.N.Z.S.P.D. Members who have become I.A.P.D. members by enrolling through their Branch will now have to deal directly with I.A.P.D. Notices inviting existing members to renew their membership will be issued a little later in the year directly by I.A.P.D. Members who wish to renew will need to deal directly with I.A.P.D.

Alistair Devlin

THE SOUTH AUSTRALIAN BRANCH...

The South Australian Branch of ANZSPD had a quiet year in 2008 with meetings suspended for 12 months. However, we have made a big come back in 2009 with well attended meetings and a lot of enthusiasm. Our most recent dinner meeting was called "The Ortho/ Paedo Interface". The format of the evening involved two paediatric dentists and two orthodontist discussing cases in pairs. Candy Mason (Paediatric Dentist) and Steve Farrer (Orthodontist) presented "A Clinical Perspective of Crowding in the Mixed Dentition. An Orthodontic-Paediatric Interface".

Early referral and a multi-disciplinary approach to management of crowding in the young patient allows the best possible outcome for the child. It is widely accepted that a crowded dentition is of multifactorial origin, with both environmental and genetic factors playing a role. Children with no anterior spacing or crowding in primary dentition have a far greater chance of crowding in the permanent dentition.

We selected three children with moderate and severe crowding to present at the meeting. Each of the children were referred for an orthodontic consultation in the early mixed dentition phase with anterior maxillary and mandibular crowding, inadequate space for lateral incisors to erupt into the arch, crossbite or ectopic eruption of first permanent molars and early loss of second primary molars.

Key points discussed included:

- Early extraction of primary teeth can be useful in guiding permanent teeth into occlusion
- Guidelines for early extraction of permanent teeth should include a sound Class I occlusion
- Early banding for arch expansion in cases of mild crowding allows treatment of crossbites and better development of the arch
- Following early loss of primary molars and mesial movement of first permanent molars, the clinician may have to wait for distal arch development before distilising 6's due to the potential damage to unerupted 7's.

Sue Springbett (Paediatric Dentist) and Marie Reichstein (Orthodontist) presented cases involving hypoplastic/hypomineralised molars. The term Molar Incisor Hypomineralisation is defined as "hypopmineralisation of systemic origin of 1-4 first permanent molars, frequently associated with affected incisors" (Weerheijm et al 2001). Hypoplasia on the other hand resulted from a lack of matrix formation although mineralization was normal. Hypomineralisation is a qualitative defect whereas hypoplasia is a quantitative defect. However the overall result in relation to treatment options was the same. From the Paediatric perspective the difficulties of managing these teeth relate to their hypersensitivity which often makes them difficult to anaesthetise. The management of the child can be more difficult due to the discomfort caused by the teeth and the need for repeated dental procedures . The severity of the defects varies from mild to severe. Treatment plans involving molar substitution, crowns and preventive measures were discussed. Marie presented the orthodontic options including the timing of extractions when necessary, favourable and unfavourable occlusions and the issue of the presence or absence of 8's.

Both presentations emphasised the need for a multidisciplinary approach to treatment.

In both presentations it was obvious that good communication and interaction between paediatric dentists and orthodontists will result in optimum care of the child with the best outcomes.

Also at this meeting Joe Verco gave a presentation on "The Long Q-T Syndrome". Joe gave this abstract of his presentation:

'The Paediatric Dentist is often at the front end of clinical diagnoses. LQTS is a cardiac condition that reflects electrical malfunction. 30–40% of sudden deaths in children are at the first event in "asymptomatic" patients. Undiagnosed syncope in sports can lead to deaths e.g. football, hockey, rowing.

The incidence of LQTS has increased from 1:5000 – 7000 to 1:1100 – 1300 in developed countries through diligent reporting. There are in excess of 300 variations, which may be genetically linked or potentiated by prescription medications ranging from antibiotics, bronchodilators (for asthma)to appetite suppressants and ADHD drugs.

Treatment varies from the use of beta-blockers, removal of the triggering drug or exercise, cardiac pacemaker to cardioverter defibrillator/or sympathetic nerve denervation.

The withdrawal of beta-blockers can lead to adrenergic bounce-back and create tachycardic problems in some instances.

A protocol of interested professional parties is suggested from general practitioners to physicians, cardiologists and geneticists.'

Reprinted courtesy of the Annals of the Royal Australasian College of Dental Surgeons, Volume 19, June 2008

Dental Caries: Where to from here?

David John Manton, BDSc, MDSc, FRACDS*

Dr Manton is a Senior Lecturer in and convener of the Paediatric Dentistry Programme in the School of Dental Science at the University of Melbourne.

Dental caries in the present day is a ubiquitous disease affecting a great proportion of the world's population.[1] In westernised communities, caries is becoming increasingly limited to certain disadvantaged sections of the community, with race, education and family income being significant caries related factors.[2-6] As well as the afore-mentioned factors, younger parents or single parent families have children with significantly higher caries rates.[7-9]

Dental Enamel

Dental enamel is the hardest and most highly mineralised tissue in the human body.[10] The physical characteristics of enamel provide a surface which effectively masticates food and resists the attritional and abrasive forces inherent in mastication and tooth to tooth contact.[10] Enamel is composed primarily of hydroxyapatite, fluorapatite and carbonated hydroxyapatite.[11] The distribution of the different anions throughout the crystal matrix affects the physical, diffusion and dissolution characteristics of the apatite.

Saliva

Once erupted, the teeth (primarily the enamel surfaces) are bathed in saliva and gingival cervical fluid (to a lesser extent). Saliva consists of water, proteins and glycoproteins, proteinases, mineral ions and enzymes.[12] Saliva has roles in the promotion of remineralisation and prevention of demineralisation, lubrication, digestion, taste, assistance with bolus formation, and anti-viral, anti-fungal and anti-bacterial functions.[12-14] Saliva is supersaturated with regard to hydroxyapatite, and therefore provides a protective milieu for the calcified components of the enamel.[13,15,16]

The supersaturation of saliva with regard to Ca^{2+} and PO_4^{3-} is maintained by the presence of macromolecules such as the statherin and prolene rich proteins (PRP) and to a lesser extent cystatins and histatins.[14,17] These macromolecules inhibit the precipitation of calcium phosphates by two mechanisms: (1) Adsorption onto the enamel surface covering the seeding sites for apatite crystal growth[15,17]; and (2) for statherin, the stabilisation of calcium and phosphate ions in the saliva via the binding action of the phosphorylated serine groups.[17,18] The phosphorylated macromolecules may play a duplicitous role, with the unbound precipitation inhibitors, once bound to the enamel surface, subsequently acting as calcium phosphate precipitators.[17]

Saliva is excreted at different rates (0.3 – 0.4 mL/min unstimulated; 1.0 – 3.0 mL/min stimulated) and with differing constituents depending on the presence or not of stimulatory factors.[19] Saliva stimulated by chewing and gustatory stimulation has increased Ca^{2+} (0.5 – 2.8 mmol/L unstimulated; 0.2 – 4.7 mmol/L stimulated) and PO_4^{3-} (2–22 mmol/L unstimulated; 1.5–25 mmol/L stimulated) ion concentrations and remineralises enamel sub-surface lesions.[13,20,21] Decreased salivary flow or quality slows the recovery of plaque pH after acid exposure and therefore increases caries risk.[22]

Dental Plaque and Dental Caries

Dental caries is a complex process of cyclical enamel de- and remineralisation. *Streptococcus mutans* and *Streptococcus sobrinus* are the two most putatively important bacteria involved in the initiation of enamel demineralization with *Lactobacillus caseii* assuming greater importance after initial progression of the carious lesion, the so-called 'specific plaque theory'.[23,24] Dental caries occurs due to organic acid production by mutans streptococci and lactobacilli as by-products of the metabolism of sugars, namely lactic, formic and acetic acids.[25] However, certain researchers have promulgated a mixed bacterial theory in which the previously mentioned cariogenic bacteria are but a few of several potentially cariogenic bacteria present in plaque.[26-28]

Repeated consumption of fermentable carbohydrates, especially sucrose, leads to the proportional overgrowth of cariogenic bacteria such as mutans streptococci, changes in the biofilm increasing potential for enamel mineral loss, the subsequent production of organic acids, and an amphibiotic change in the oral microflora leading to increased risk of dental caries.[23,29-31]

Frequent sucrose ingestion also leads to the production of insoluble glucans by *S. mutans* which alters the diffusion characteristics of the plaque, increasing the diffusion rate of hydronium ions (H+), and decreasing the concentration of calcium and phosphate ions.[30] These organic acids, mainly lactic, diffuse through the biofilm and into the enamel pores between the rods where they dissociate and decrease the pH of the fluid surrounding the enamel crystals.[32] The critical pH for enamel dissolution is often quoted simplistically as pH = 5.5, relating to the Stephan curve; however, the solubility of the specific calcium phosphate species relate to their degree of saturation in the peri-crystal fluid.[32] The demineralisation starts with peripheral loss of mineral from the crystals, leading to an enlargement of inter-rod spaces, allowing further diffusion of the organic acids.[24]

 * Presented at the Nineteenth Convocation of the Royal Australasian College of Dental Surgeons, Hong Kong, May-June 2008.

If the balance is tilted continually towards demineralisation the enamel will lose enough mineral to change its optical characteristics and appear whiter than the adjacent sound enamel – the white spot lesion (WSL), with the optical changes occuring due to the increased pore spaces between the thinned rods and the effect this has on the refractive qualities of the enamel.[24,33]

An individual is never free of dental caries. The process of enamel demineralisation and remineralisation is constantly cycling between net loss and gain of mineral. It is only when the balance leans towards net loss that clinically identifiable signs of the process become apparent. The long-term outcome of this cycling is determined by the composition and extent of plaque, sugar consumption frequency and timing, fluoride exposure, salivary flow and quality, enamel quality and immune response.[31,32,34] In summary, the disease is manifested as interplay between environmental, behavioural and genetic factors.[23]

Fluoride

The use of fluoride has been the primary method for the prevention of dental caries and erosion for the past 60 years.[35] The fluoridation of public water supplies has been rated as one of the most significant public health measures in the last century; however there are limitations to this method of fluoride delivery, as there is a reduction in the progression of caries but little reduction in incidence.[32,36,37]

The fluoride ion acts mainly by driving remineralisation due to solubility differences between fluorapatite and hydroxyapatite and by decreasing enamel solubility in two ways: (1) the fluoride ion is more stable in the crystal lattice than the hydroxyl ion, and (2) it interacts closely with the calcium ions on the crystal surface, binding them strongly in the matrix.[38] At the pH levels commonly found in actively fermenting dental plaque, fluorapatite may be supersaturated, whereas hydroxyapatite is undersaturated, leading to the preferential deposition of the less soluble fluorapatite.[39]

The pre-eminent role of fluoride in preventive dentistry remains valid; however the effectiveness of fluoride to remineralise enamel and obtain net mineral gain is limited by the levels of bio-available Ca^{2+} and PO_4^{3-} [32,40,41] Therefore if the acid challenge to the enamel is great, the salivary Ca^{2+} and PO_4^{3-} reservoir is quickly depleted and net loss of enamel mineral can occur.[42]

The intrinsic sources of Ca^{2+} and PO_4^{3-} are from saliva, dissolved tooth structure and to a lesser degree, gingival crevicular fluid, and therefore to gain net remineralisation the action of fluoride is limited by the amount of salivary Ca^{2+} that is bioavailable.[13,42,43] Increased concentrations of Ca^{2+} would also increase the retention of fluoride in the plaque biofilm by increasing calcium-bridging.[44] Therefore, the supplementation of oral bioavailable calcium and phosphate has the potential to significantly decrease enamel demineralisation and increase remineralisation.

Casein Phosphopeptide-Amorphous Calcium Phosphate Nanocomplexes

Ongoing research by the Reynolds group over the past 25 years has isolated and purified the peptides from casein that have the major anti-cariogenic effect – the casein phosphopeptides (CPP) containing the amino acid sequence Ser(P)-Ser(P)-Ser(P)-Glu-Glu. Casein phosphopeptide-amorphous calcium phosphate nano-complexes* (CPP-ACP) have been demonstrated to have anti-cariogenic activity in laboratory, animal and human *in situ* and clinical experiments.[21,41,45-47]

The CPPs have the ability to stabilise Ca^{2+} and PO_4^{3-} in metastable solution.[48-50] Through the multiple phosphoseryl residues, the CPPs bind to forming nano-clusters of calcium and phosphate preventing the growth of seed crystals to the critical size required for nucleation and phase transformation, providing a ready source of ionic calcium and phosphate.[48]

The nanocomplexes are bound in plaque and buffer the Ca^{2+} and PO_4^{3-} activities in the plaque fluid at the tooth surface, establishing an environment supersaturated with calcium and phosphate, inhibiting demineralisation and driving remineralisation.[50] The CPPs also bind strongly to the surfaces of *Streptococcus mutans*, with the CPP bond being twice the strength of that of calcium and therefore provide a source of calcium and phosphate in the plaque fluid and also slowing the diffusion and subsequent loss of calcium from the plaque fluid.[51,52] The adherence of *S. mutans* and *S. sobrinus* within dental plaque and onto hydroxyapatite is also reduced significantly by CPP-ACP, decreasing the proportion of the mutans streptococci in rats and in *in vitro* studies.[53-55]

A number of recent studies have reported the efficacy of CPP-ACP in the *in vitro* inhibition of enamel and dentine demineralisation,[45,56-58] and also the promotion of *in vitro* remineralisation of enamel subsurface lesions (ESL).[43] When tested using an in situ model, similar results have been reported, with CPP-ACP driving remineralisation of ESL with delivery by chewing gum, mouthrinse, crème or lozenge.[21,46,50,59] Several case reports show the remineralising and desensitizing effects of CPP-ACP, and a recent randomised controlled clinical trial supported the caries preventive and remineralising effects of casein phosphopeptides.[60-64]

When CPP-ACP is delivered in conjunction with fluoride (CPP-ACFP), the potentiating effect of fluoride on enamel remineralisation is significantly increased.[41,43] Interestingly, the fluoride is not concentrated in the outer 'hypermineralised' surface layer as normally reported, but detected throughout the depth of CPP-ACP/F remineralized ESLs.[41,43] The proposed mechanism is that the CPP stabilises the Ca^{2+}, PO_4^{3-}, and F^- ions at the enamel surface in the correct molar ratios and allows the ions to diffuse into the ESL down concentration gradients subsequently forming fluorapatite.[41,43] Remineralisation of ESLs is greatest when CPP-ACP and CPP-ACFP are delivered at pH 5.5.[41]

* Recaldent™, CASRN 691364-49-5

Non-Peptide Based Remineralising Technologies

ACP®† – this product is based on the mixing of two solutions, one containing phosphate ions ($K_4P_2O_7$) and the other calcium ions ($CaNO_3$), after which there is precipitation of calcium and phosphate salts, including hydroxyapatite on the surface of the tooth.

Enamelon®‡ – this product is based on sodium fluoride and ionic calcium and phosphate, putatively providing a precipitate layer of fluor/hydroxy-apatite after use.

Novamin®§ – this product is a bioactive glass calcium sodium phosphosilicate comprising: 45% SiO_2, 24.5% Na_2O, 24.5% CaO and 6% P_2O_5. The calcium and phosphate ions are released from the bioactive glass on contact with salivary fluids and a precipitate layer of calcium phosphate, most likely carbonated hydroxyapatite is formed on the seeding surface.

The lack of a stabilising agent to prevent precipitation of the calcium phosphate salts after saturation concentrations of the ions are reached is a major disadvantage of these technologies. This therefore limits the availability of ionic calcium and phosphate at the tooth surface, subsequently limiting remineralisation. Due to the lack of calcium and phosphate ion stabilisation, these technologies are limited to forming an aggregation of a calcium phosphate salt precipitate on the tooth surface. This mechanism has been reported to decrease dentinal sensitivity by physically blocking exposed tubules, however potassium nitrate is a byproduct of the formation of the amorphous calcium phosphate, therefore the sole or additive effect of potassium nitrate in decreasing sensitivity cannot be discounted.[65] All of these calcium phosphate technologies are, however, limited in their effectiveness for desensitisation, as they require constant use of the product, as the precipitate can be removed by attrition and dissolution during normal oral function.

In summary, caries still persists as a major health problem 60 years after the widespread introduction of fluoridated products. A new technology (CPP-ACP; Recaldent™)* utilising and enhancing the important role of calcium and phosphate in the prevention and remineralisation of caries has been developed recently. CPP-ACP has three major mechanisms of decreasing caries activity: (1) by inhibiting demineralisation, (2) by promoting remineralisation, and (3) by increasing the acid resistance of the remineralised enamel. These mechanisms are facilitated by the bioavailability of supersaturated supplies of Ca^{2+} and PO_4^{3-} stabilised by the CPP and potentiate the preventive and remineralising effects of fluoride.

References

1. Petersen PE, Bourgeois D, Ogawa H, Estupinan-Day S, Ndyaie C. The global burden of oral diseases and risks to oral health. Bulletin of the World Health Organization 2005;83:661-69.
2. Armfield JM. Socioeconomic Inequalities in Child Oral Health: A Comparison of Discrete and Composite Area-Based Measures. J Pub H'lth Dent 2007;67:119-25.
3. Brennan DS, Roberts-Thompson KF, Spencer AJ. Oral Health of Indigenous adult public dental patients in Australia. Aust Dent J 2008;52:322-28.
4. Australian Research Centre for Population Oral Health. Dental visiting among the Australian adult dental population. Aust Dent J 2008;52:336-39.
5. Marshall TA, Eichenberger-Gilmore JM, Broffitt BA, Warren JJ, Levy SM. Dental caries and childhood obesity: roles of diet and socioeconomic status. Comm Dent Oral Epidemiol 2007;35:449-58.
6. Psoter WJ, Pendrys DG, Morse DE, Zhang H, Mayne ST. Associations of Ethnicity/Race and Socioeconomic Status with Early Childhood Caries Patterns. J Pub Hlth Dent 2006;66:23-29.
7. Marshall TA, Eichenberger-Gilmore JM, Larson MA, Warren JJ, Levy SM. Comparison of the intakes of sugars by young children with and without dental caries experience. J Am Dent Ass 2007;138:39-46.
8. Armfield JM, Spencer AJ. Consumption of nonpublic water: implications for children's caries experience. Community Dent Oral Epidemiol 2004;32:283-96.
9. Dye BA, Shenkin JD, Ogden CL, Marshall TA, Levy SM, Kanellis MJ. The relationship between healthful eating practices and dental caries in children aged 2-5 years in the United States, 1988-1994. J Am Dent Ass 2004;135:55-66.
10. Avery JK. Enamel. In: Steele PF, editor. Essentials of Oral histology and Embryology. 2nd ed. St Louis, Missouri, USA: Mosby Inc.; 2000. p. 84-93.
11. Mkhonto D, de Leeuw NH. A computer modelling study of the effect of water on the surface structure and morphology of fluorapatite: introducing a Ca-10(PO4)(6)F-2 potential model. J Mat Chem 2002;12:2633-42.
12. Fox P. Salivary enhancement therapies. Caries Res 2004;38:241-46.
13. Edgar W, Dawes C, O'Mullane D. Saliva and Oral Health. 3rd ed. London: British Dental Association; 2004. p. 120-28.
14. Huq NL, Cross KJ, Ung M, Myroforidis H, Veith PD, Chen D, et al. A Review of the Salivary Proteome and Peptidome and Salivary-derived Peptide Therapeutics. Int J Peptide Res Ther 2007; 13:547-64.
15. Moreno EC, Varughese K, Hay DI. Effect of Human Salivary Proteins on the Precipitation Kinetics of Calcium Phosphate. Calcif Tissue Int 1979;28:7-16.
16. Larsen MJ. Degrees of saturation with respect to apatites in parotid saliva at various pH values. Scand J Dent Res 1975;83:7-12.
17. Cross KJ, Huq NL, O'Brien-Simpson NM, Perich JW, Attard TJ, Reynolds EC. The role of Multiphosphorylated Peptides in Mineralized Tissue Regeneration. Int J Peptide Res Ther 2007; 13:479-95.
18. Huq NL, Cross KJ, Ung M, Reynolds EC. A review of protein structure and gene organisation for proteins associated with mineralised tissue and calcium phosphate stabilisation encoded on human chromosome 4. Arch Oral Biol 2005;50:599-609.
19. Edgar M, Dawes C, O'Mullane D. Saliva and Oral Health. British Dental Association. 3rd ed. London; 2004. p. 44-49.
20. Dawes C, Kubieniec K. The effects of prolonged gum chewing on salivary flow rate and composition. Arch Oral Biol 2004;49:665-69.
21. Cai F, Manton DJ, Shen P, Walker GD, Cross KJ, Yuan Y, et al. Effect of addition of citric acid and casein phosphopeptide-amorphous calcium phosphate to a sugar-free chewing gum on enamel remineralization in situ. Caries Res 2007;41:377-83.
22. Johansson A-K, Lingstrom P, Birkhed D. Effect of soft drinks on proximal plaque pH at normal and low salivary secretion rates. Acta Odont Scand 2007;65:352 - 56.
23. Fejerskov O. Changing paradigms in concepts on dental caries: consequences for oral health care. Caries Res 2004;38:182-91.
24. Kidd EAM, Fejerskov O. What constitutes dental caries? Histopathology of carious enamel and dentin related to the action of cariogenic biofilms. J Dent

† American Dental Association Foundation, Chicago, IL, USA
‡ Enamelon Inc., Cranbury, NJ, USA
§ Novamin Technology Inc., Gainesville, FL, USA

Res 2004;83 (Spec Iss C):C35-C38.

25. Geddes DA. Diet patterns and caries. Adv Dent Res 1994;8:221-4.
26. Lingstrom P, van Ruyven FO, van Houte J, Kent R. The pH of dental plaque in its relation to early enamel caries and dental plaque flora in humans. J Dent Res 2000;79:770-77.
27. Kleinberg I. A mixed-bacteria ecological approach to understanding the role of oral bacteria in dental caries causation: an alternative to Stretococcus mutans and the specific plaque hypotthesis. Crit Rev Oral BiolMed 2002;13:108-25.
28. Beighton D. The complex oral microflora of high-risk individuals and groups and its role in the caries process. Comm Dent Oral Epidem 2005;33:248-55.
29. Ruby J, Goldner M. Nature of symbiosis in oral disease. J Dent Res 2007;86:8-11.
30. Leme AFP, Koo H, Bellato CM, Bedi G, Cury JA. The Role of Sucrose in Cariogenic Dental Biofilm Formation--New Insight. J Dent Res 2006;85:878-87.
31. Vale GC, Tabchoury CPM, Arthur RA, Del Bel Cury AA, Leme AFP, Cury JA. Temporal relationship between sucrose-associated changes in dental biofilm composition and enamel demineralization. Caries Res 2007;41:406-12.
32. Aoba T. Solubility properties of human tooth mineral and pathogenesis of dental caries. Oral Dis 2004;10:249-57.
33. Robinson C, Shore RC, Brookes SJ, Strafford S, Wood SR, Kirkham J. The chemistry of enamel caries. Crit Rev Oral Biol Med 2000;11:481-95.
34. Featherstone JDB. The continuum of dental caries--evidence for a dynamic disease process. J Dent Res 2004;83 Spec No C:C39-C42.
35. Jones S, Burt BA, Petersen PE, Lennon MA. The effective use of fluorides in public health. Bulletin of the World Health Organization 2005;89:670-76.
36. Morbidity and Mortality Weekly Report. Fluoridation of Drinking Water to Prevent Dental Caries. In: SERVICES USDOHH, editor: Centre for Disease Control; 1999. p. 933-40.
37. Fejerskov O, Ekstrand J, Burt BA. Fluoride in dentistry. Copenhagen: Munksgaard; 1996.
38. de Leeuw NH. Resisting the onset of hydroxyapatite dissolution through the incorporation of fluoride. J Phys Chem B 2004;108:1809-11.
39. ten Cate JM. Current concepts on the theories of the mechanism of action of fluoride. Acta Odontol Scand 1999;57:325-9.
40. Featherstone JD. Caries prevention and reversal based on the caries balance. Pediat Dent 2006;28:128-32.
41. Cochrane NJ, Saranathan S, Cai F, Cross KJ, Reynolds EC. Enamel subsurface lesion remineralisation with casein phopshopeptide stabilised solutions of calcium, phosphate and fluoride. Caries Res 2008;42:88–97.
42. Yamazaki H, Litman A, Margolis HC. Effect of fluoride on artificial caries lesion progression and repair in human enamel: Regulation of mineral deposition and dissolution under in vivo-like conditions. Arch Oral Biol 2007;52:110-20.
43. Reynolds EC, Cai F, Cochrane NJ, Walker GD, Morgan MV, Reynolds C. Fluoride and casein phosphopeptide-amorphous calcium phosphate. J Dent Res 2008;87:344-48
44. Kato K, Nakagaki H, Arai K, Pearce EI. The influence of salivary variables on fluoride retention in dental plaque exposed to a mineral-enriching solution. Caries Res 2002;36:58-63.
45. Sudjalim TR, Woods MG, Manton DJ, Reynolds EC. Prevention of demineralization around orthodontic brackets in vitro. Am J Orthodont Dentofac Orthoped 2007;131:e1-705.e9.
46. Cai F, Shen P, Morgan MV, Reynolds EC. Remineralization of enamel subsurface lesions in situ by sugar-free lozenges containing casein phosphopeptide-amorphous calcium phosphate. Aust Dent J 2003;48:240-43.
47. Manton DJ, Walker GD, Cai F, Cochrane NJ, Shen P, Reynolds EC. Remineralization of enamel subsurface lesions in situ by the use of three commercially available sugar-free gums. Int J Paediatr Dent 2008 Apr 21. [Epub ahead of print] PMID: 18435723.
48. Reynolds EC. Anticariogenic complexes of amorphous calcium phosphate stabilized by casein phosphopeptides: a review. J Spec Care Dent 1998;18:8-16.
49. Cross KJ, Huq NL, Palamara JE, Perich JW, Reynolds EC. Physicochemical characterization of casein phosphopeptide-amorphous calcium phosphate nanocomplexes. J Biol Chem 2005;280:15362-69.
50. Reynolds EC, Cai F, Shen P, Walker GD. Retention in plaque and remineralization of enamel lesions by various forms of calcium in a mouthrinse or sugar-free chewing gum. J Dent Res 2003;82:206-11.
51. Rose RK. Binding characteristics of Streptococcus mutans for calcium and casein phosphopeptide. Caries Res 2000;34(5).
52. Rose RK. Effects of an anticariogenic casein phosphopeptide on calcium diffusion in streptococcal model dental plaques. Arch Oral Biol 2000;45:569-75.
53. Schupbach P, Neeser JR, Golliard M, Rouvet M, Guggenheim B. Incorporation of caseinoglycomacropeptide and caseinophosphopeptide into the salivary pellicle inhibits adherence of mutans streptococci. J Dent Res 1996;75:1779-88.
54. Guggenheim B, Schmid R, Aeschlimann JM, Berrocal R, Neeser JR. Powdered milk micellar casein prevents oral colonization by Streptococcus sobrinus and dental caries in rats: a basis for the caries-protective effect of dairy products. Caries Res 1999;33:446-54.
55. Neeser JR, Golliard M, Woltz A, Rouvet M, Dillmann ML. In vitro modulation of oral bacterial adhesion to saliva-coated hydroxyapatite beads by milk casein derivatives. Oral Microbiol Immunol1994;9:193-201.
56. Yamaguchi K, Miyazaki M, Takamizawa T, Inage H, Kurokawa H. Ultrasonic determination of the effect of casein phosphopeptide-amorphous calcium phosphate paste on the demineralization of bovine dentin. Caries Res 2007;41:204-07.
57. Yamaguchi K, Miyazaki M, Takamizawa T, Inage H, Moore BK. Effect of CPP-ACP paste on mechanical properties of bovine enamel as determined by an ultrasonic device. J Dent 2006;34:230-36.
58. Rahiotis C, Vougiouklakis G. Effect of a CPP-ACP agent on the demineralization and remineralization of dentine in vitro. J Dent 2007;35:695-98.
59. Manton DJ, Shen P, Cai F, Cochrane NJ, Reynolds C, Messer LB, et al. Remineralisation of White Spot Lesions in situ by Tooth Mousse. J Dent Res 2006;85(Spec Iss B):185.
60. Vlacic J, Meyers IA, Walsh LJ. Combined CPP-ACP and photoactivated disinfection (PAD) therapy in arresting root surface caries: a case report. Br Dent J 2007;203:457-9.
61. Ng F, Manton DJ. Aesthetic management of severely fluorosed incisors in an adolescent female. Aust Dent J 2007;52:243-8.
62. Milnar FJ. Considering biomodification and remineralization techniques as adjuncts to vital tooth-bleaching regimens. Compend Contin Educ Dent 2007;28:234-6, 38-40.
63. Ardu S, Castioni NV, Benbachir N, Krejci I. Minimally invasive treatment of white spot enamel lesions. Quintessence Int 2007;38:633-6.
64. Morgan MV, Adams GG, Bailey DL, Tsao CE, Fischman SL, Reynolds EC. The Anticariogenic Effect of Sugar-Free Gum Containing CPP-ACP Nanocomplexes on Approximal Caries Determined Using Digital Bitewing Radiography. Caries Res 2008;42:171–84.
65. Giniger M, Macdonald J, Ziemba S, Felix H. The clinical performance of professionally dispensed bleaching gel with added amorphous calcium phosphate. J Am Dent Assoc 2005;136:383-92.

Address for correspondence:

School of Dental Science

The University of Melbourne

720 Swanston Street

Carlton Victoria 3053.

djmanton@unimelb.edu.au

ANZSPD Convention 2010

Join us at the Heritage Hotel in Queenstown on 29 and 30 March 2010 for a stimulating scientific programme and exciting social events in one of the most beautiful areas of New Zealand. Thanks again to the generous sponsorship from Colgate as the principal sponsor of the convention, we are thrilled to confirm Professor John Featherstone (School of Dentistry, UCSF, San Francisco) as our keynote speaker, along with Dr Yasmi Crystal and a number of sought-after speakers from New Zealand and Australia. A number of topics relating to clinical and academic paediatric dentistry will be covered, with a central focus on the overall health and wellbeing of our children and adolescents.

Preparations are also underway for an outstanding social programme for delegates, their partners and children encompassing a range of options from adventure sports (bungy anyone?), or dancing to arguably New Zealand's best covers band, to relaxing with an outstanding glass of Central Otago wine.

Why not take an extended Easter break and make the most of your opportunity to visit the region? Queenstown is at it's most beautiful in the autumn – we look forward to welcoming you.

Colgate PRINCIPAL SPONSOR Colgate

Klocking Up the Ks

Dr Kylie Pearce

Hello everyone,

I am sure that you all heard about the plane crash in Papua New Guinea where several Australians lost their lives. Two fantastic people from the Sunshine Coast were tragically killed before they were able to trek the Kokoda trail. Dr June Canavan was a local sports medicine physician and she had taken up a quest to conquer 5 treks all starting with "K" to raise money for the School of St Jude in Tanzania. Dr Canavan's project was called Klocking up the K's, and aimed to raise much needed funds for the School of St Jude, run by an Australian woman in Tanzania. A friend, Keith Gracie had joined her for the Kokoda leg of the quest.

Tragically Dr June and Keith were killed before their dream was realised. I have travelled throughout the world, and recognise poverty in all areas. For those of you who don't know me, I am a paediatric dentist, and my views are that children can only reach their full potential if they are given every possible opportunity to succeed. Without basic education these children would never know what life could offer. I spend a lot of time travelling to the more "remote" and underprivileged areas of the world, and I am always fascinated by the way the children are forced to live their lives for their families. All too often education is set aside, and it is hard to think that a lot of children never get to reach their full potential. The School of St Jude in Tanzania identifies the brightest and poorest children, and helps to give them an education. Without the generosity of Gemma Sisia these children would not have an education.

As I am about to climb Kilimanjaro at the end of September (29 September to 5 October) I felt it was my duty to do the climb for this wonderful organisation. When I return to Australia I will than be completing the Melbourne half marathon on 11 October. If you think that I can achieve these two targets then please donate to this wonderful cause.

I am thrilled to be climbing on behalf of the Klocking up the K's charity, and although they have achieved the $50 000 target that Dr June set, the charity work is going to continue and I hope to be able to do more in the future. If you have any other questions or would like to make a donation please email me at kylieclimbskili@optusnet.com.au.

Kylie

Colgate Corner

by Dr Barbara Shearer
Scientific Affairs Manager

barbara_shearer@colpal.com

16th Biennial Convention of the Australian and New Zealand Society of Paediatric Dentistry

Queenstown

28-30 March 2010

Colgate will once-again act as the Principal sponsor of ANZSPD's biennial convention in Queenstown early next year. There is an excellent line-up of speakers and I know the conference committee will be working very hard to ensure a great social program. I heard John Featherstone speak earlier this year and was impressed with his evidence-based approach to caries risk assessment. I am sure this will be a very valuable session for everyone in clinical practice.

I hope that many of you will be able to attend and take advantage of the great location. The Colgate team in NZ is looking forward to welcoming you and demonstrating our product range.

Senior Dental Leaders Program

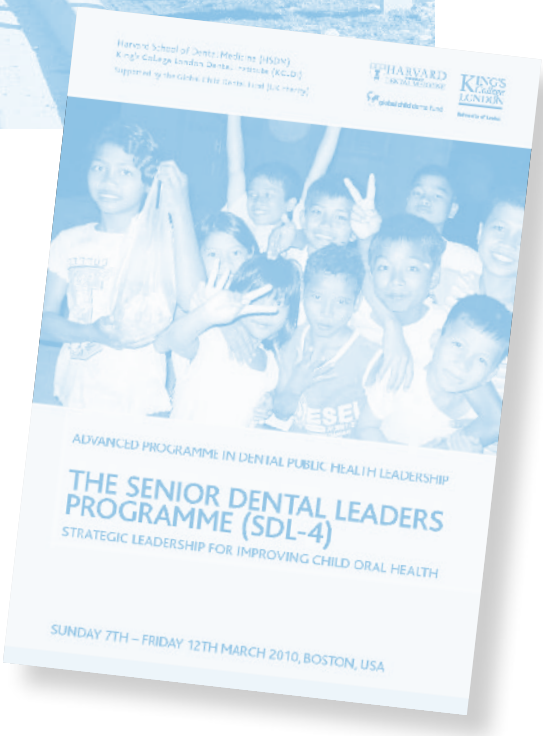
Boston 2010

In addition to the donation of 100,000 brush/paste kits per year, Colgate also contributes to the Australasian Global Child Dental Health Taskforce by supporting an ANZ representative at the annual Senior Dental Leaders program. In 2010 the course will be held in Boston, 7-12 March. The program is designed to bring together in a global network, individuals who wish to engage at a local, federal or global level in bringing about lasting positive change in the area of child dental health.

Colgate is proud to support the ANZ representative by covering the US$10,000 course fee and accommodation costs.

Further information and an application form can be obtained by emailing Dr Barbara Shearer (Barbara_Shearer@colpal.com)

Applications close 13 November 2009.



Colgate Territory Managers are here to assist you with the products you need in your surgeries

NSW: Sophie Hughes 0448 421 699 Starley Marshall 0419 993 700 Janne Thomas 0410 488 581
QLD: Anna Bagnell 0409 159 417 Lisa Lowther 0417 642 665 | ACT: Debbie Goodwin 0419 268 549
VIC: Natasha Dostal 0458 280 739 Melissa Wicker 0437 499 511 Catherine Byriell 0417 598 170 | WA: Angela Tascone 0400 505 223
SA/NT: Leanne Nelson 0400 387 249 | NZ: Glenda McKenzie 64 21 621 315 Debra Reardon 64 21 593 986 Lisa Grant 64 21 593 985

Colgate Sales Managers ACT/NSW/QLD: Nolene Devery 0419 998 515 | SA/TAS/VIC/WA: Janine Vincent 0417 592 499

Orders for Colgate products are placed through Henry Schein Halas/Shalfoon Australia 1300 658 822 (F) 1300 658 810 | NZ: 0800 808 855 (F) (09) 3781 158

Coming events

4 – 7 November 2009
Australasian Osseointegration Society 7th Biennial Conference

Gold Coast Convention and Exhibition Centre, Queensland, Australia
For further information, please contact the conference managers:
Phone: +61 7 3858 5525
Fax: +61 7 3858 5499
Email: info@aosconference.com.au
Website: www.aosconference.com.au

28 November 2009
NZ Branch of ANZSPD Annual Study Day

Macs Brewery
Wellington, New Zealand

28-30 March 2010
16th Biennial Convention of ANZSPD

Queenstown, New Zealand
www.conference.co.nz.anzspd2010

28 March 2010
Australasian Academy of Paediatric Dentistry Biennial Meeting

Skyline and Gondola
Queenstown, New Zealand

27-31 May 2010
63rd AAPD Annual Session

Chicago, Ill

11-13 June 2010
16th World Congress Dental Traumatology

Verona, Italy

14-17 July 2010
88th IADR General Session and Exhibition

Barcelona

26-29 May 2011
64th AAPD Annual Session

New York, NY

Australia and New Zeaand Society of Paediatric Dentistry
www.anzspd.org.au

Federal President — Dr Nina Vasan, nvasan@xtra.co.nz

Federal Secretary Manager — Dr Alistair Devlin, devlins@iinet.net.au

Branch Executives

Branch	President	Secretary	Fed Councillor
NSW	Dr Philippa Sawyer philippa.sawyer@toothdoctor.net.au	Dr Suzanne Brent suzannebrent@yahoo.com.au	Dr Kareen Mekertichian ksmek@bigpond.net.au
QLD	Dr Steve Kazoullis steven@kazoullis.com	Daniel Ford ford@pdgdental.com.au	Dr John Rutar john.rutar@bigond.com
SA	Dr PJW Verco	Dr Sue Springbett drsue@childrensdentistry.com.au	Dr PJW Verco
VIC	Dr Margarita Silva	Fiona Ng prodontome@iinet.net.au	Dr John Sheahan
WA	Dr Kate Dyson	Dr Alistair Devlin devlins@iinet.net.au	Dr Tim Johnston
NZ	Dr Erin Mahoney	Craig Waterhouse craig.shona@xtra.co.nz	Dr Nina Vasan

Editor Synopses — Dr Dorothy Boyd, djboyd@xtra.co.nz

Correspondence — Dorothy Boyd
The Editor, Synopses
Burns House Dental Specialists
Level 7, 10 George Street
Dunedin, New Zealand 9010

Printing and distribution — Colgate

Colgate Oral Care
Level 15, 345 George Street
Sydney NSW 2000 AUSTRALIA

Mailing List

The mailing list for the distribution of Synopses is maintained by Dr John Winters on behalf of the Federal Secretary/Manager of ANZSPD. It is compiled from information supplied by the Branch Secretaries. If there are errors in your mailing details, please contact Dr John Winters or your Branch Secretary. Please do not contact Colgate for address correction.

Submissions

All text for inclusion in Synopses must be submitted to the editor on floppy disk, zip disk, CD or by email. Both PC and MAC formats are accepted. Media will not be returned. Address email to djboyd@xtra.co.nz. Please enclose your contact details and email address with all submissions.

Deadline next issue 20 January 2010